Ines Mandl, Ph.D.
Columbia University College of Physicians and Surgeons
630 West 168th Street
New York, New York 10032

Elastolytic Breakdown in the Etiology of Pulmonary Emphysema. Grant #546-AR1

The main emphasis of this study will be placed on establishing the etiology of pulmonary emphysema by experiments designed to adduce evidence for our working hypothesis that excessive availability of elastolytic enzymes causes damage to lung elastin which leads to loss of elasticity. In familial panlobular emphysema a deficiency gene has been found responsible for low titers of serum inhibitors for trypsin, elastase and other proteolytic enzymes. Patients with this condition are being screened as well as their relatives and the identify of the factors involved will be ascertained. At the same time the specific enzyme, probably leucocyte in origin, which is inhibited in normal individuals and free to act in the emphysema patients is sought. An animal model has been established in rats which are made emphysematous by injection of elastolytic enzymes. Induction of the disease can be prevented by progesterone injections. The mechanism leading to the establishment or the prevention of the disease will be explored further. Lung tissue obtained from autopsy specimen and from experimental animals will be studied with special emphasis on the changes in composition of elastin from normal and emphysematous persons and/or animals.

Current Grant Level: $33,875.

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Source: https://www.industrydocuments.ucsf.edu/docs/mxl0000